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SOME POINTS OF SPECIAL INTEREST IN THE STUDY OF THE DEEP REFLEXES OF THE LOWER EXTREMITIES.

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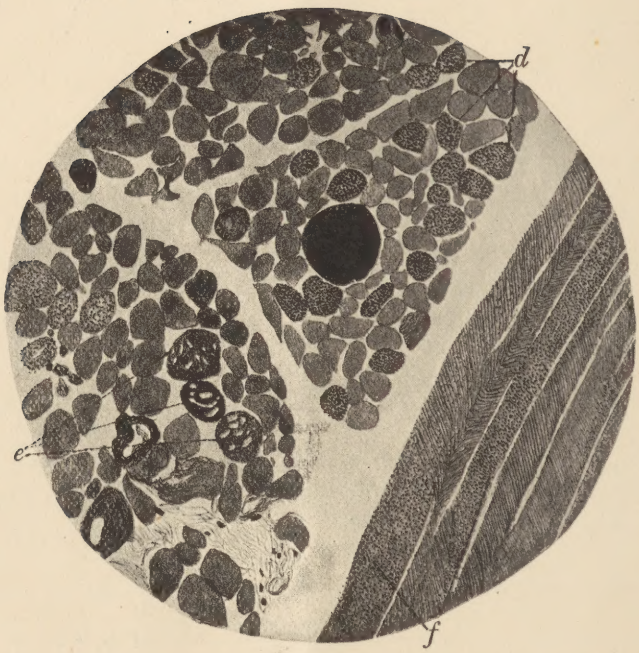

FIG. III. On the left of the drawing is a transverse section of muscular fibers stained by Marchi's method; on the right a longitudinal section stained by the same method. Many fibers are filled with numerous black dots (*d f*) representing fatty degeneration of the muscular fibers. Some of the fibers show vacuoles (*e*). In the center of the picture is a large fiber that has undergone hyaline degeneration.

SOME POINTS OF SPECIAL INTEREST IN THE STUDY OF THE DEEP REFLEXES OF THE LOWER EXTREMITIES.[1]

1. ANKLE-CLONUS WITH ABSENCE OF KNEE-JERK; 2. THE SIGNIFICANCE OF ANKLE-CLONUS IN THE DIAGNOSIS OF HYSTERIA FROM ORGANIC DISEASE; 3. PATELLAR CLONUS; 4. TENDO-ACHILLIS JERK IN TABETICS.

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ANKLE-CLONUS WITH ABSENCE OF KNEE-JERK.

The patient, H. J., white, laborer, was admitted to the men's wards of the Philadelphia Hospital, September 12, 1898. On admission he was found to be paralyzed in both legs. He had great difficulty in breathing, complained of pain in the precordial region and of a band of pain encircling the body between the ensiform cartilage and the umbilicus. His family history showed nothing of importance. When fifteen he had had rheumatism, but otherwise up to the time of his present illness he had been healthy. He admitted the moderate use of both alcohol and tobacco.

About five weeks before his admission to the hospital he began to be troubled with constipation and difficulty in expelling his urine. The bladder would become distended and painful, and it was only with great difficulty that he could urinate. One week previous to his admission he began to have a feeling of numbness in both feet, this gradually extend-

[1] Read before the New York Neurological Society, Jan. 3, 1899.

ing upwards. Five days before admission he went to bed with no evidence of paralysis, but on attempting to rise the following morning he found that his legs were paralyzed, the precordial and girdling pain developing about the same time, while breathing became so difficult that he had to assume a sitting position.

Examination on admission showed him to be a man of good muscular development, but with an appearance of being poorly nourished. His face had a waxy look, lips and finger tips were bluish and cold, and dyspnea was marked. His tongue protruded straight without tremor; it was pale and anemic looking, tooth-marked at the edges and coated with a dirty white fur. His pulse was rapid, small and water-hammer in character.

The normal heart sound was nowhere heard. At the mitral area two loudly blowing murmurs were heard, the louder being systolic and transmitted toward the axilla. At the aortic cartilage two more murmurs were heard; the one, diastolic, was soft and blowing; the other, systolic, was harsh and rasping. The latter was plainly transmitted into the vessels of the neck. Pressure over the upper part of the abdomen caused pain.

When admitted the foot of the right leg could be lifted about six inches from the bed when the leg was extended, but in the left this movement could not be carried out, and when it was attempted the leg flexed at the knee and the heel dragged. The toes of both feet moved freely. When catheterized immediately after admission the urethra was found to be hyperesthetic, and while passing the catheter both legs and thighs were quite forcibly flexed, whether voluntarily or because of reflex action was not definitely determined. It was learned that the patient had had no headache, that his appetite had been very poor, and for a few months he had had some cough. The pupils were equal and reacted both to light and in accommodation, and no paralysis of any of the extraocular muscles was present at the time of admission or later. At no time had he any paralysis or anesthesia of the upper extremities.

He was examined by me three hours after admission. He did not, and apparently could not, make any movements with the right leg, which seemed to be completely paralyzed, and was semi-flexed at the hip and knee. On the anterior, inner and outer aspect of both knees and on the dorsum of the foot were dusky reddish areas.

Examination for sensation showed that the patient was analgesic over the right thigh and front of the leg below the knee, and on the dorsum of the foot to its inner edge. Tactile sensation was preserved, as was also the thermal sense. In

general terms the sensory phenomena in the left lower extremity were the same in distribution as in the right, but a little less extended over the dorsum of the foot and great toe. A glance at the diagram (Fig. I) gives a correct idea of the extent of the analgesia, and a comparion of this with one of Heiberg's plates (Fig. II) shows that the areas chiefly affected were those supplied by the anterior crural, the communicans peronei, and the musculocutaneous or superficial peroneal nerve.

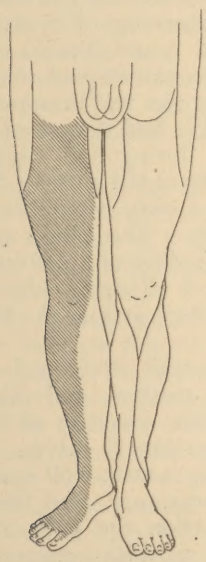


FIG. I.

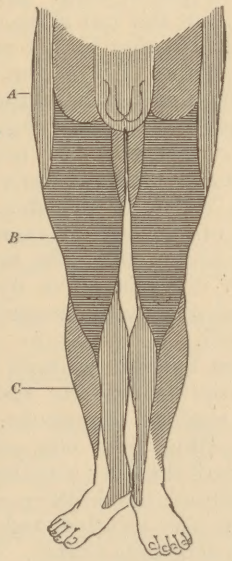


FIG. II.

FIGURE I. Area of analgesia in the patient with lost knee-jerk on both sides and ankle-clonus on one side.

FIGURE II. Sensory nerve areas of the anterior surface of the lower extremity: A, external cutaneous (cutaneus femoris lateralis); B, crural; C, external saphenous (communicans peronei); musculocutaneous (unshaded area on dorsum of foot).

The most interesting clinical point in connection with the examination of this man was that brought out by a study of the muscle and tendon phenomena in the lower extremities. Quadriceps-jerk on each side was prompt and marked; a striking fact in connection with the intense degeneration of the muscle subsequently found, and additional evidence that the muscle-jerk is essentially different from the knee-jerk. Both knee-jerks, for which he was frequently tested, were absent. Ankle-clonus was undoubtedly present on the left side; some-

times it was quickly exhausted, but at other times it persisted; it was never absent at any of the examinations which were repeated several times during three days. Ankle-clonus and toe-jerk were both absent on the right side, and toe-jerk was not present on the left. Both elbow-jerks were absent.

Most of the clinical phenomena above noticed were determined on September 12, the day of the patient's admission to the hospital. At 7 P. M. of this day he seemed to be worse and complained of severe pain in the small of the back and over the precordium. He passed a comparatively comfortable night, but did not sleep any. On the morning of September 13, he said that he felt much better and would like to sit up in his chair. The action of his heart was slower and steadier, and the bluish color of the lips and finger tips had disappeared. The color of his face was also better. He had one stool containing mucus. He slept quite well during the night of September 13th, but early on the morning of the 14th he had a slight attack of dyspnea. He passed a very uncomfortable night on the 14th; dyspnea was extreme. The action of the heart was forcible and rapid, notwithstanding the continuous use of digitalis. The dyspnea continued all day. He complained greatly of precordial pain. He died at 3.40 A. M. on the 15th.

The autopsy showed the body of a well developed man. Post-mortem rigidity and lividity were marked, and the feet were slightly edematous. A considerable quantity of clear, light yellow fluid was present in the peritoneal cavity. The surface of the liver was smooth, and its edges were well marked. On section the knife met with little resistance, and the cut section was mottled and fatty. On section the pulp of the spleen was quite friable. The suprarenal capsules were normal. The right and left kidneys showed similar conditions. The fatty capsule was thick; the fibrous capsule stripped with difficulty, leaving a rough surface in areas. On section the cortex was found to be narrowed. The substance of the kidneys was quite firm. The right and left pleural cavities contained about 500 c. c. of fluid.

The left lung on section showed marked edema and at its apex a slight consolidation. It also showed a slight anthracosis. Similar conditions existed in the right lung. Much subpericardial fat was present. The heart muscle, particularly around the left ventricle, and more especially the papillary muscles, showed marked fatty change. Both the aortic and mitral valves were sclerotic, the aortic admitting two fingers and the mitral four.

Careful examination of every part of the brain showed no gross lesion. Considerable fluid blood was found in the

vertebral canal external to the dura, but not more than is often seen in spinal autopsies. No organized clots were found at the level of any of the lumbar segments or elsewhere in the canal. The spinal membranes were not thickened and showed no signs of either diffuse or focal meningitis. No gross lesion of any of the spinal nerves in their intraspinal course could be detected.

The entire spinal cord was put in formalin for hardening. Portions of one of the posterior spinal roots from the first lumbar segment, and also parts of the anterior crural nerve on each side were removed, and placed in a one per cent. solution of osmic acid. Other portions of the anterior crural nerves were placed in Müller's fluid. The pieces of nerve were taken from just below Poupart's ligament. A fragment of muscle was taken from the same region and was put in Müller's fluid.

Segments from the first lumbar region and lower levels of the spinal cord were embedded in celloidin, cut and stained by the methods of v. Lenhossék and Weigert, by acid fuchsin, and by Delafield's hematoxylin. Sections were also stained by Marchi's method.

The microscopical examination of the specimens was made in the William Pepper Laboratory of Clinical Medicine, of the University of Pennsylvania, by Dr. William G. Spiller, who has furnished the following report:

"The sections from the spinal cord stained by the above methods appear to be perfectly normal. The cells of the anterior horns are as numerous as in normal cords, and stained by the method of v. Lenhossék show the usual arrangement of the chromophilic elements and the central position of the nucleus. No traces of hemorrhage can be found in the lumbar region, and the vessels are not distended to an unusual degree. The anterior and posterior spinal roots stained by acid fuchsin and the hematoxylin of Weigert seem to be normal—that the posterior, however, are not *absolutely* normal is shown by the osmic acid. No degeneration of the lateral columns can be detected by the method of Weigert or of Marchi.

"A portion of one posterior root taken from the first lumbar segment and stained by osmic acid shows many fibers in which the myelin has assumed a beaded form, and probably more such fibers than a normal posterior root contains; this root, therefore, cannot be said to be *perfectly* normal, and yet the changes are perhaps too unimportant to explain any of the symptoms.

"Portions of one of the anterior crural nerves stained with osmic acid and "teased" present many fibers in which the

myelin has become irregularly beaded and others in which the myelin stains faintly with osmic acid.

"Sections from the anterior crural nerves stained with acid fuchsin and Delafield's hematoxylin appear to be nearly normal, each nerve fiber containing an axis cylinder, but when the hematoxylin of Weigert is employed certain bundles seem to contain fewer nerve fibers than is normal, and also an unusual number of small nerve fibers. These sections were made at a considerable distance from the peripheral terminations of the nerves.

"Section from the piece of muscle removed present a high degree of alteration of muscular tissue. When stained by Marchi's method, which is most valuable for recent degeneration of muscle, numerous fibers are found both in longitudinal and transverse sections filled with an excessive number of small black dots representing a fatty degeneration (Fig. III). In transverse sections the numerous rounded, greatly swollen muscular fibers, that have undergone hyaline degeneration and vacuolation, are most striking. Some of these fibers are very large, perfectly round, stain a deep red with acid fuchsin, and present a glassy appearance (Fig. IV). When stained by Van Gieson's method these swollen fibers are of a dark wine color. The sarcolemma nuclei have in general disappeared from those muscular fibers that are entirely degenerated. The interstitial connective tissue nuclei are not very excessive. The blood vessels of the muscles are normal. Intramuscular nerve bundles contain fibers each with an axis cylinder when the acid fuchsin is used, and by the hematoxylin of Weigert the medullary sheaths are stained black, although possibly less intensely so than in normal muscles

"In longitudinal section the blocks of hyaline degenerated muscles are exceedingly numerous, and quite frequently one is able to trace one of these swollen blocks without a trace of transverse striation, into a normal fiber transversely striated containing sarcolemma nuclei, and of much smaller caliber than the hyaline block in which it originates (Fig. V). In some of these swollen hyaline muscles the interior is more faintly stained that the exterior. Numerous spaces in certain of these hyaline fibers resemble vacuoles. The absence of proliferation of the connective tissue and of atrophied muscular fibers is very striking, and the process was evidently a recent one. Many of these hyaline blocks in longitudinal sections present longitudinal striation, and in some the peculiar arrangement of the hyaline tissue in irregular bands is seen, as described by Marinesco."

The findings in this case of lost knee-jerks with presence of ankle-clonus may be briefly stated. The spinal cord appears

to be perfectly normal; a posterior root from the first lumbar segment stained with osmic acid exhibits a somewhat unusual number of nerve fibers with beaded myelin; the anterior crural nerves seem to be distinctly degenerated when the osmic acid is employed, but the changes are slight as shown by Weigert's hematoxylin and the acid fuchsin; the muscular tissue is greatly altered and many of the muscular fibers are tumefied and present a high degree of hyaline or fatty degeneration.

In the light of the clinical history and pathological findings, what is the most probable explanation of the unusual syndrome—absent knee-jerk with ankle-clonus on one side—presented by this case? During the patient's life the most probable diagnosis seemed to be a focal lesion involving the lumbar cord in that region through which the reflex arc for the patella is completed, namely, the lumbar segments from the second to the fourth inclusive. One objection to this diagnosis was, however, to be found in the distribution of the analgesia, which was not confined to the area supplied by the crural nerve, derived from the lumbar cord and plexus, but also included the areas for the communicans peronei and musculocutaneous (superficial peroneal of Heiberg), which are branches of the external popliteal, and, therefore, derived from the sacral cord and plexus. The existence of heart disease with both mitral and aortic murmurs lent color to the view that the lesion might be one or more foci of softening in the cord. Another theory that seemed tenable was that a lesion like a hemorrhage involved some of the extramedullary or intramedullary lumbar roots, or the cells of the anterior horns, and compressed the lateral columns of the cord in the upper or midlumbar region, but against this view was the improbability of a localized hemorrhage causing the irregular distribution of the analgesia, although, of course, an irregular clot, an exudate, or multiple clots might produce almost any unusual grouping of symptoms. As has been stated in the history of the autopsy and microscopical examination, the most critical investigation revealed no lesion of the substance of the cord or of the intramedullary nerve roots in the lumbar region, though one of the first lumbar posterior roots examined was not entirely normal. The blood found in the

vertebral canal was fluid and only such as is frequently seen in spinal autopsies, the leakage from the tissues wounded in removing the posterior portions of the vertebral column. Fluid blood could not have produced the symptoms, for gravity would have caused the blood to descend to the lower part of the spinal column, and ankle-clonus would then hardly have been obtained.

The findings in this case give us an explanation for absent knee-jerk and presence of foot-clonus not hitherto afforded, in fact I know of no similar case with necropsy. The muscular tissue examined was greatly altered, presenting marked hyaline and fatty degeneration, while the anterior crural nerves were partially degenerated, as was also the posterior root examined. I attribute the lost knee-jerks conjointly to disease of the muscles and to the degeneration of the crural nerves, alterations in muscle and nerve tissue affording a comparatively easy explanation of the loss of the patellar reflexes.

It is not improbable that the difficulty in voiding the urine and feces, of which the patient complained, was due to a degeneration of the muscles of the abdominal walls, similar to that in the piece of sartorius examined, as the contraction of the abdominal muscles plays an important part in the evacuation of bladder and rectum. I cannot regret sufficiently that muscle and nerve tissue were not taken from widely separated portions of the body and submitted to careful microscopical examination. The symptoms presented by the patient were those which most neurologists would attribute to disease of the spinal cord.

We must seek to find the cause of this muscular degeneration, which was of an intense degree in the piece of sartorius examined. It seems not improbable that the cardiac trouble may have been the cause. The absence of atrophied fibers and of proliferation of the connective tissue, and the presence of recent degeneration, as shown by the method of Marchi, are in conformity with the short duration of the paralysis in the lower limbs. We know that muscles undergo hyaline degeneration as a result of acute infectious disease, but I have been unable to obtain the history of any such affection in my patient. Muscular degeneration may be more common

than we suppose when the heart has been acting feebly for a long time.

This is the day for the theory of autointoxication. It may be that we have to seek the cause of the muscular degeneration of this case in the imperfect elimination of toxic substances from the system, brought about by an impaired circulation. Whatever theory is preferred, the fact must be accepted that the sartorius muscle of my patient presented signs of intense degeneration, probably of recent origin, and that the degeneration of the muscles was apparently much greater than that of the nerve fibers. We should be cautious, however, in accepting a degeneration of nerve fibers which appears slight under the microscope, as of little importance. Nerve cells and nerve fibers probably suffer great impairment of function before they undergo structural change, and apparently slight degeneration of nerve tissue may cause severe functional disturbance.

The explanation of the distinct and persistent ankle-clonus on one side is not easy, indeed remains somewhat in doubt, and is worthy of discussion.

While it is well known that in multiple neuritis the tendon reflexes are often absent at some stage, and especially early in this disease, exaggerated knee-jerks have been observed. Usually as the disease advances the reflexes diminish, and in the majority of cases disappear. Even ankle-clonus has been observed in rare instances in multiple neuritis.

In one case of multiple neuritis, with the symptoms of a comparatively mild form of erythromelalgia, I have even observed slight patellar clonus on one side at one of the examinations, but this could not be elicited subsequently. This was a man twenty-five years old, studied in my wards at the Philadelphia Hospital, three weeks after the onset of the disease.

What is most significant in its bearings on the case under discussion is that v. Bechterew[3] in one case of multiple neuritis observed ankle-clonus after the patellar reflex had disappeared.

With these facts before us it is possible that the unilateral

[3] v. Bechterew, W., Neurologisches Centralblatt, 1895, p. 1157.

ankle-clonus present in my patient was due to a neuritis attacking the nerve branches to the tendo Achillis. In multiple neuritis the degree of inflammation and degeneration may vary at different times in different nerve trunks and branches, and it is probable that if inflammation was present in this case in the nerves to the tendo Achillis the disease was here in an early stage.

It may be that the muscles and nerves of the calf were not involved in the degenerative process, and the absence of analgesia over the posterior and inner aspects of the leg makes this view not improbable.

Some cases of lost knee-jerk with presence of ankle-clonus in which inflammation of peripheral nerves is seen may be explained on the theory that the cells of the sacral region controlling the ankle phenomenon are thrown into a state of hyperexcitability by the inflammation of nerves having their reflex arc in the lumbar segments concerned with the patellar reflex.

Another explanation is to be found in the fact that while it is uncommon to find exaggerated reflexes in neuritis, it has been shown by Weir Mitchell[4] that certain forms of irritation of a nerve trunk may cause excessive irritability in the muscles supplied by the affected nerve, as was seen when a frozen nerve was thawed.

It is scarcely necessary to an audience of neurologists to direct attention to the anatomy and physiology of the knee-jerk and of ankle-clonus, and yet it may be of some little service to do this as a part of the discussion of this unusual pathological grouping of the two most important of the deep reflexes of the lower extremities, especially as it is not yet firmly established through which segments of the lumbar and sacral cord these reflexes are consummated. In the knee phenomenon the tendon of the patella having been struck, the excitation is carried to the spinal cord by crural sensory fibers, and enters by the dorsal roots into a certain portion of the dorsal column, and thence passes to the dorsal horn; next it takes its course through the intermediate gray substance until it reaches the cells of the ventral horn; and thence

[4] Mitchell, S. W., Injuries of Nerves and Their Consequences, p. 60.

the motor excitation goes through the motor roots and crural nerve to the anterior muscles of the thigh. It does not matter for the purposes of our discussion whether knee-jerk is or is not a pure reflex, or whether, as is most probable, it is due first to direct stimulation of the muscle, and secondly to reflex influence; in any case absence of this phenomenon follows a complete break in any portion of this path. In considering the loss of knee-jerk in this as in any other case, therefore, it should be remembered that it may be abolished from disease of any of the following parts: the peripheral sensory nerve fibers, the posterior roots, either in their extramedullary or intramedullary portion, the gray matter through which the reflex collaterals of the posterior roots pass, the cells of the ventral horns, the motor roots, the motor nerve fibers, or the extensor muscles of the leg.

According to Westphal's investigations, when the patellar reflex was abolished the part of the spinal cord affected was in the transitional region from the thoracic to the lumbar cord. Many later researches have placed the spinal segments for this reflex at a lower position of the cord, and according to different observers it would seem that the reflex arc might be situated anywhere from the second to the fifth lumbar segment.

Pineles[5], in describing a case which he designates as one of sacral tabes with lost knee-jerks, says the first normal fibers of the posterior roots, in examining the cord from below upward, were observed in the midlumbar region, and from here upward the root entrance zones were gradually filled with normal fibers. Redlich[6], says that the patellar reflex arc is now believed to be a little lower than Westphal placed it, and that usually it is located about the second lumbar segment. Another case of sacral tabes has recently been reported by Auerbach[7]. The knee-jerk was absent on each side. The degeneration by microscopical examination seemed to be in the first and second sacral and the lower lumbar posterior roots.

[5] Pineles, Arbeiten aus dem Institut für Anatomie und Physiologie des Centralnervensystems an der Wiener Universität, Heft IV., p. 341.

[6] Redlich, E., Die Pathologie der tabischen Hinterstrangserkrankung, p. 100.

[7] Auerbach, Deutsche Zeitschrift für Nervenheilkunde, V. XI., p. 143.

Higher up the posterior root fibers were normal. In the middle portion of the lumbar region the posterior roots were practically intact. This case is therefore similar to the one reported by Pineles.

In a case of tumor of the cauda equina, reported by Dejerine and Spiller[8], knee-jerks were lost, and all the posterior roots were degenerated as high as, but not including, the first lumbar roots. The knee reflex must be lower than the first lumbar, as indicated by this case, for the examination was made by the method of Marchi, which would have revealed any recent degeneration in the first lumbar roots if it had been present, and did reveal intense degeneration of the roots below the first lumbar.

From these and other investigations it is at least clear that the segments of the spinal cord included in the patellar reflex arc are situated somewhere between the second and fifth lumbar segments inclusive. The facts before us would seem to indicate that it is probably located where it has been placed by Edinger[9], Starr[10] and others, namely, in the second or third lumbar segments, or both.

The anatomy of the Achilles reflex arc has not been so thoroughly studied as that of the patellar reflex arc; in fact I have not at command any investigations definitely locating the segments of the sacral cord concerned in this reflex. Some clinical and anatomical considerations would, however, indicate that the segment of the cord concerned in this reflex is probably situated where it has been placed by Starr and others, namely, somewhere from the first to the third sacral segments inclusive. Paralleling the description above given of the patellar reflex arc and its lesions, it would therefore follow that loss of the Achilles reflex would occur from a destructive lesion affecting the peripheral sensory nerve fibers from the tendo Achillis, the dorsal roots of these fibers, the gray matter

[8] Dejerine and Spiller, Comptes rendus de la Soc. de Biologie, 1895, and personal communication by Dr. Spiller.

[9] Edinger, L., Vorlesungen über den Bau der nervösen Centralorgane des Menschen und der Thiere, fünfte Auflage, Leipzig, 1896, p. 313-314.

[10] Starr, M. A., The Diagnosis and Localization of Spinal Cord Diseases, in A System of Practical Medicine by American Authors, ed. by Loomis and Thompson, V. 4, 1898, p. 71.

through which the reflex collaterals of these roots pass, the cells of the ventral horns, the motor roots, the motor nerve fibers to the gastrocnemius and soleus muscles, or these muscles themselves.

It is not enough to know the particular segment or segments of the cord concerned in the patellar reflex. Only certain portions of these segments are involved in the reflex arc, and it therefore follows that lesions affecting the spinal cord at the proper *level* to include the reflex arc might, if limited to certain transverse regions of the cord, permit the reflex act to be consummated. Pineles[11] says that Westphal's thorough investigations have clearly shown that when the patellar reflex is absent a certain zone in the spinal cord must be diseased. This zone is limited by an imaginary line parallel to the posterior septum, drawn through the point where the posterior horn makes a bend, by the inner side of the posterior horn and by the periphery of the cord. This zone was called by Westphal the root-entrance zone (Wurzeleintrittszone). Westphal's investigations referred only to the transitional region from the thoracic to the lumbar cord, but it is probable that an area in one or more of the lumbar segments somewhere from the second to the fourth inclusive, corresponding to Westphal's zone, contains the patellar reflex arc. A lesion outside of this area, and outside of the other portions of the spinal cord included in the reflex arc, might be present without any disturbance of the patellar reflex. A focal lesion like a hemorrhage or small focus of softening, or a disease like syringomyelia, might involve the segments of the cord at the level which corresponds to the patellar reflex arc without abrogating the patellar phenomenon, a fact which must be borne in mind in the explanation of some exceptional cases of lumbar focal disease. Such a case of retained patellar reflex in a case of tabes, for example, has recently been reported by Achard and Lévi.[12] The root-entrance zones were comparatively intact, although the posterior columns were much degenerated.

[11] Pineles, Arbeiten aus dem Institut für Anatomie und Physiologie des Centralnervensystems an der Wiener Universität, Heft IV, p. 341.

[12] Achard, C., and Lévi, L., Nouvelle iconographie de la Salpêtrière, 2, 1898, p. 83.

In order to show at a glance the mechanism of the patellar and the ankle phenomena I have had made a diagram which shows both the patellar reflex arc and the Achilles reflex arc,

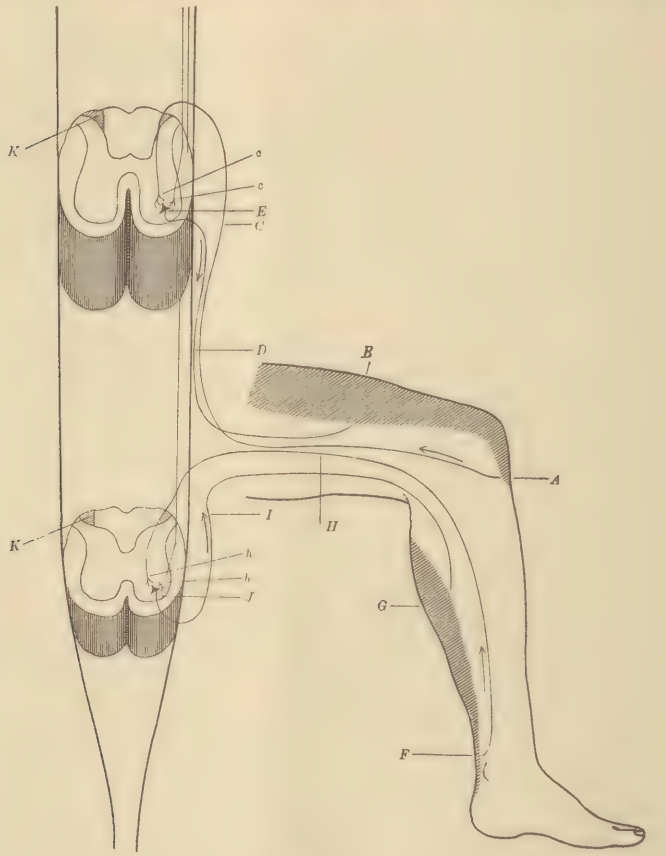


FIG. VI.

FIGURE VI. Scheme of the patellar and the tendo-Achillis reflex arc: A, patellar tendon; B, quadriceps muscle; C, sensory (afferent) fiber from the patellar tendon; D, motor fiber from cell in the ventral horn to quadricep muscle; E, cell-body in ventral horn; F, tendo Achillis; G, gastrocnemius muscle; H, sensory fiber from the tendo Achillis; I, motor fiber passing from cell in the ventral horn to gastrocnemius muscle; J, cell-body in ventral horn; K, area corresponding to Westphal's zone; the direction of the arrows shows the direction of the sensory and motor impulses.

and the controlling tracts related to these arcs. A brief study of this diagram (Fig. VI) will show that a destructive lesion in-

volving the lumbar segment or segments in which the patellar reflex arc is included might abrogate the patellar reflex on both sides, and at the same time by involving the corticospinal tract to the sacral cord remove the cerebral inhibition from the Achilles reflex and thus allow the foot phenomenon to coexist with lost knee-jerk. A glance at this diagram will also show how advanced disease involving the nerve and the muscle forming portions of the patellar reflex arc would destroy the patellar reflex, the Achilles reflex arc remaining, and this perhaps by less advanced and irritative nerve disease being stimulated until foot-clonus is manifested. It will also be seen from a study of this diagram that limited focal disease of the cerebral cortex, or of the cerebrospinal tract, might cause increased clonus on one side, while peripheral disease of crural nerves or quadriceps muscle might destroy the patellar reflex. Other possible explanations which will be given of lost knee-jerk with ankle-clonus can also be better understood by a study of the diagram.

Let me next glance at the literature of the subject of lost knee-jerk with presence of ankle-clonus. This is limited. I have been able to find the record of only ten cases, although, of course, it is not improbable that some have been overlooked. In Sternberg's[18] monograph reference is made to seven cases. I have notes of three other cases, which, including my own, make eleven in all. The cases being so few, a summary of the most important of them will, I am sure, not be considered out of place. Bechterew (l. c.), as already indicated, in one case of multiple neuritis has observed ankle-clonus after the patellar reflex had disappeared.

Dr. F. X. Dercum, in a personal communication to the writer, has given the details of the case of a patient who, after falling from a height, striking on his buttocks, became paraplegic. At the time of examination this patient could flex his thighs slightly on his abdomen, but had no control over his feet. Marked wasting of some of the muscles of the lower extremities was present, and the man had blebs or blisters over his toes, ankles and feet. He made considerable improvement

[18] Sternberg, M., Die Sehnenreflexe und ihre Bedeutung für die Pathologie des Nervensystems, Leipzig und Wien, 1893.

while under Dr. Dercum's care, but remained partially paraplegic. He presented no evidence of local injury to the spinal column. The conditions as to sensation were not recalled by Dr. Dercum. In this case knee-jerk was lost on both sides and ankle-clonus was present on both sides.

Erb[14] has recorded a case in which the reflex clonus and the biceps tendon reflex were observed, but the patellar reflex and the reflex of the adductors was in no way to be obtained. This was one of three cases of compression of the spinal cord with kyphosis. The kyphosis was in the lumbar region, the first and second lumbar vertebræ forming the point of the kyphosis.

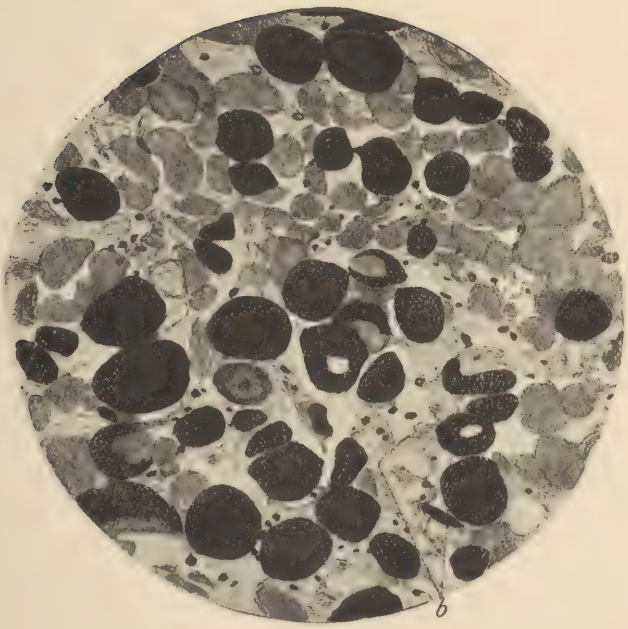
It will be recalled by members of the New York Neurological Society that Dr. Joseph Fraenkel[15], at the March meeting of this year, presented a child two and a half years old, in whom the knee-jerks were absent and ankle-clonus was present on both sides. The child, born by the breech after a difficult labor, had abnormal lower extremities from birth, and was not, at the time of presentation to the society, able to sit, walk, or stand. Electrical examinations showed extensive degeneration of all the muscles except those of the calves. Dr. Fraenkel seemed, from his remarks, to incline to the view that the case was one of poliomyelitis, or that the syndrome was the result of a dropsy, a focal hemorrhage, or a localized cavity formation. He explained the existence of the ankle-clonus by the theory that the calf-muscles were in a state of increased tonus, due to the fact that the antagonists were gone. Dr. Joseph Collins accepted the pathological explanation given by Dr. Fraenkel, but Dr. B. Sachs believed it better to assume that in the case there was some developmental defect.

At the meeting of the Berlin Society for Psychiatry and Nervous Diseases, held November 8, 1886, Mendel[16] presented a man of forty with (1) loss of patellar reflex; (2) bilateral foot-clonus; and (3) paradoxical contraction in the left foot. This patient, who was not syphilitic, began in 1877 to have pains in the left hip, which varied in intensity and later were felt in the

[14] Erb, W. Archiv. f. Psychiatrie, 5, p. 802.

[15] Fraenkel, J., Proceedings of the New York Neurological Society, Jour. Nerv. and Ment. Dis., V. 25, April, 1898.

[16] Mendel, E., Archiv. für Psychiatrie, V. 19, pp. 524-525.



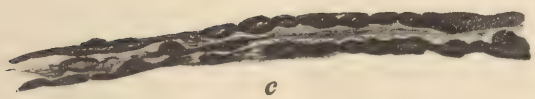


FIG. IV. Transverse section of the sartorius muscle stained with acid fuchsin. The small unaltered fibers measure about 30 microns, while the larger ones that have undergone hyaline degeneration (represented in black in the drawing) measure 70 to 100 microns. Some of the fibers show vacuoles (*b*). (*c*) Degenerated fibers from one of the anterior crural nerves. The myelin sheaths are irregularly beaded.

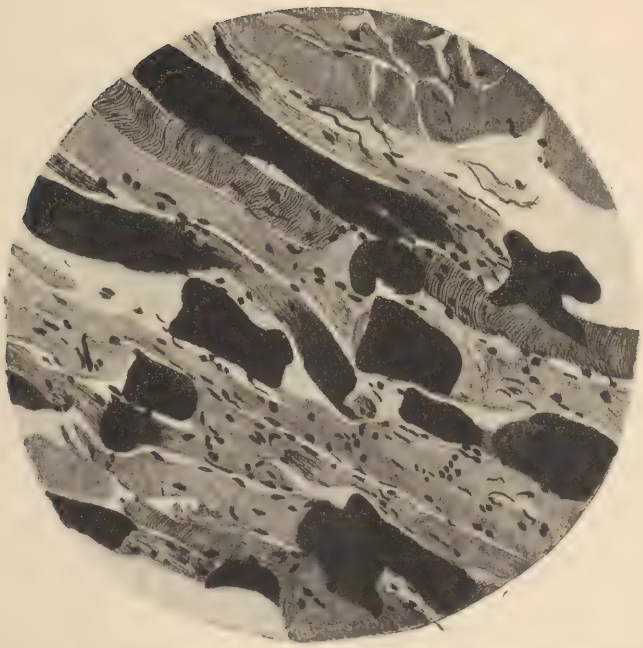


FIG. V. Longitudinal section of the sartorius muscle The hyaline masses which were round when viewed in transverse section (Fig. IV.) are long and irregular in Fig. V. In many muscular fibers the transverse striation is lost, though in some it is preserved. Some muscular fibers terminate in hyaline masses.

right leg and shoulder. The legs became weaker while the arms remained intact. In the lower limbs considerable disturbance of cutaneous sensation was found and the muscle sense was impaired. The muscles of the thigh and calf were atrophic, especially on the left side. The electrical reactions could not be taken. The left pupil was larger than the right. The left half of the face was not as well innervated as the right. Mendel believed the case was one of multiple sclerosis, and that a focus was located in the reflex arc of the quadriceps muscle; that another was more deeply seated, and in the lateral column, involving the sciatic (control) tract. One of these foci caused loss of the knee-jerk, the other caused foot-clonus. Remak had examined the electrical conditions of this patient previously, and found a great diminution in the response of the left crural nerve, indications of reaction of degeneration in the quadriceps extensor and no contraction of the anterior tibial muscle from irritation of the left peroneal nerve. He believed that a circumscribed transverse myelitis was present in the upper part of the lumbar swelling, and that this on the left side had involved the cells and caused paralysis in the crural territory, with implication of the anterior tibial. Oppenheim said he had often seen cases with dorsal clonus of the foot with Westphal's sign (loss of knee-jerk), for example, in a case of vertebral injury. It seems to me probable that Oppenheim may not have fully considered his words when he made the sweeping assertion that he had *often* seen foot-clonus with lost knee-jerk.

In connection with Mendel's explanation of the syndrome in this case it may be remarked that Buzzard[17], in a discussion on the clinical significance of the deep reflexes before the Medical Society of London, stated that the occurrence of ankle-clonus in a leg which presented at the same time a total absence of knee-reflex, not infrequently pointed to a disseminated sclerosis.

Fleury[18], in a case of typhoid fever examined on the twelfth day of the disease, found the tendon reflexes (presum-

[17] Buzzard, Brit. Med. Jour., Nov. 7, 1885, V. 2, p. 868.

[18] Fleury, Note sur les rapports de la trépidation épiletoide du pied avec l'exagération des réflexes rotuliens, in Revue de Médecine, quatrième année, 1884, V. 4, Paris, p. 656.

ably from the context the patellar reflexes) absolutely abolished. While this was the case, he obtained on the contrary at the first attempt a very marked trepidation in the right as well as in the left foot. The tendon reflexes remained abolished during the entire time that the patient was in the hospital. The trepidation also continued, but presented notable variations. Fleury concluded that epileptoid trepidation does not always coexist with exaggeration of the patellar reflexes, and that it can exist in subjects in whom these reflexes are normal, impaired or abolished.

Regarding the probable lesions in a case which presents the unusual syndrome of lost knee-jerk with presence of ankle clonus, several conclusions may be drawn from a study of the case here presented and an examination of the meager literature of the subject:

(1) The syndrome may be due to a compression or destroying lesion, such as caries with pachymeningitis, or transverse myelitis, involving the spinal cord in the region of the patellar reflex arc, namely, somewhere between the second and fifth lumbar segments, and most probably about the second or third lumbar segments.

(2) The syndrome may be due to disseminated sclerosis, foci of sclerosis being present both in the reflex arc for the patella and in the lateral column.

(3) The syndrome may be due to focal lesions like hemorrhage, softening, or cavity formation, attacking points in the reflex arc and also the lateral columns.

(4) The syndrome may be due to peculiar forms of developmental arrest of the spinal cord, as for instance, to defect in the gray matter of the lumbar segments and in the lateral columns.

(5) The syndrome may be due to a combination of muscular and neural disease, as in my case, and as was probably also the case in the man suffering from typhoid fever as recorded by Fleury. It is known that hyaline degeneration of muscular fibers occurs especially in typhoid fever. Fleury's case was probably in its pathology not unlike my own.

(6) On theoretical grounds it seems probable that the syndrome might be due to a focal lesion in the cerebral cortex, or in the cortical spinal (pyramidal) tract, or to arrested de-

velopment of the tract, associated with disease (inflammation or degeneration) limited to the crural nerves and their muscles.

THE SIGNIFICANCE OF ANKLE-CLONUS IN THE DIAGNOSIS OF ORGANIC FROM FUNCTIONAL DISEASES, ESPECIALLY HYSTERIA.

The second subject connected with the study of the deep reflexes of the lower extremities to which, in accordance with the announcement of this meeting, it is my purpose to direct attention is one which some of those present may at first sight regard as so far settled as not to be worthy of special discussion; that this, however, is not the case is forced upon me by several considerations. The discussion of this subject at the Philadelphia Neurological Society has brought out a striking difference of opinion among neurologists of large experience, a fact familiar to those who have read the proceedings in the JOURNAL OF NERVOUS AND MENTAL DISEASE.[19] I have not much to present in addition to what has already been published in these proceedings, but believing the subject to be one still worthy of close discussion I have concluded to include its presentation in this paper, hoping to get decided expressions of opinion from the members of the New York Neurological Society.

The majority of neurologists, both in this country and abroad, so far as I have been able to note their opinions, seem to disagree, in part at least, with Gowers and the writer regarding ankle-clonus in hysteria and other functional affections. v. Bechterew[20] says that many neurologists still share the opinion of Gowers, although cases of pure hysteria with ankle-clonus have been observed. Bechterew says also that he has seen ankle-clonus, exactly like that occurring in organic disease, in cases of myoclonus multiplex, and also he has seen it in cases of akinesia algera. Oppenheim[21] believes that both foot-clonus and paradoxical contraction occur in hysteria.

[19] Proceedings of the Phila. Neurological Society, Jour. Nerv. and Ment. Dis., v. 24, Nov., 1897.

[20] Bechterew, W., Neurologisches Centralblatt, 1895, p. 1157.

[21] Oppenheim, H., Archiv. für Psychiatrie, v. 19, pp. 524-525. (In the discussion of a paper presented by Mendel to the Berlin Society for Psychiatry and Nervous Diseases, Nov. 8, 1886.)

Sternberg,[22] in his monograph on the tendon reflexes, says that in about twenty per cent. of hysterical cases foot-clonus is present.

As my own views are practically in accord with those of Gowers,[23] let me recall these. While not denying the possibility of a true uniform clonus in hysterical paraplegia, Gowers holds that it is so rare that it does not materially lessen the value of this sign of organic disease. "Certain facts," he says, "might be stated with confidence. 1. In many cases the myotatic irritability was perfectly normal. 2. In others there was distinct, though slight excess, insufficient to give a true clonus. 3. In hysterical contracture, and depending on it, there might be a clonus like that which occurred in health in standing on 'tip-toe.' 4. A 'spurious foot clonus' was common, depending on a voluntary contraction in the calf muscles, pressing down the foot, and varying in degree from time to time, the clonus varying with it. This was very characteristic and was a most important diagnostic sign of hysterical paraplegia.

"Apart from the slight clonus produced through a voluntary depression of the foot of the patient in response to the passive flexion of the ankle, and readily recognized, and the true clonus which may be obtainable during hysterical contracture, a true foot clonus or a rectus clonus deserves the greatest weight, as all but conclusive evidence of organic disease. I have known many mistakes in diagnosis," he says, "in which lateral sclerosis was mistaken for hysterical paraplegia owing to disregard of the evidence afforded by this symptom, but I have never known the opposite error from undue regard to this symptom. Moreover, an excess of myotatic irritability in so-called hysterical paralysis must depend on more than functional disease. There must be changes in nutrition, and consequent persistent defective control of the muscle reflex centers."

What I believe may be justly designated a large clinical experience, extending over many years, leads me to subscribe fully to the views thus expressed.

[22] Sternberg, M., Die Sehnenreflexe und ihre Bedeutung für die Pathologie des Nervensystems, p. 254.

[23] Gowers, Sir W. R., Brit. Med. Jour., Nov. 7, 1885, v. 2; and A Manual of Diseases of the Nervous System, v. 1, 2d ed., Phila., 1892, p. 451.

If I am right in following Gowers in these expressions of opinion, the most important sources of error in those who maintain the contrary are (1) the nonrecognition of the coexistence of organic lesions, and especially focal lesions and hysteria in the same case; (2) the misinterpretation of cases in which toxemia or malnutrition are more important factors than hysteria; and (3) absolute errors of diagnosis.

The fact that organic disease, and especially a focal lesion, either cerebral or spinal, is often associated with hysteria, and sometimes even induces grave hysterical manifestations, may mislead even the skilful diagnostician with regard to ankle-clonus. In the cases reported to the Philadelphia Neurological Society by Spiller[24] and by Burr, this combination of organic disease and hysteria seemed to me a source of error. In Spiller's case the patient was paralyzed in his right arm and to a less degree in the right leg, had intention tremor in the right limbs, sensation on the right side had been partially lost but had been regained, knee-jerk and quadriceps-jerk were exaggerated in the right lower extremity, positive and persistent ankle-clonus was present on the right side only, other tendon and muscle phenomena were also exaggerated on the right side, chin-jerk was present, and some amnesia for language was an interesting feature. Most of those who took part in the discussion of this case leaned to the view that it was one of hysteria, although to my mind it was undoubtedly one of organic hemiplegia. The disappearance of the anesthesia was not inconsistent with the view that it may have been a pressure phenomenon due to a lesion of the posterior limb of the left internal capsule. The double hemiplegia may have been apparent rather than real, and even the history so carefully given is susceptible of other explanation than that of hysteria. At the most, I believe that a critical study of the entire case shows it to be one of grave organic disease probably associated with grave hysteria. In the case recorded by Burr,[25] the patient was a hemiplegic of a somewhat common motor type, with great

[24] Spiller, W. G., An Unusual Case of Hemiplegia, Jour, Nerv. and Ment. Dis., v. 24, No. 7, July, 1897.

[25] Burr, C. W., A Case of Hemiplegia (Possibly Hysteric) with Ankle-Clonus, in the Proceedings of the Philadelphia Neurological Society, Jour. of Nerv. and Ment. Dis., v. 24, No. 11, Nov., 1897, p. 707.

exaggeration of all the reflexes, including persistent ankle-clonus; but in addition, this man had a number of attacks which seemed to be clearly hysterical with motor, emotional and sensory manifestations of a marked character. At times he had complete left hemianesthesia, at other times anesthesia of the segmental type. The case was probably organic, with hysterical epiphenomena. Seguin[26] believed that hysterical phenomena were much more frequent in left than in right hemiplegics.

With regard to the misinterpretation of cases in which toxemia or pernicious malnutrition plays a part, I would only say that the diagnosis of pure hysteria is the matter of vital importance. Hysteria of pure and typical form is a disease already so large in its manifestations as not to require us to attribute to it symptoms due to other causes. As held by Gowers, profound nutritional disorders may be present in hysteria as in organic disease, but the symptoms due to these nutritional changes are not in a strict sense a part of the hysteria. A patient suffering from syphilis or from alcoholism may have either hysteria or neurasthenia, but it is syphilis with neurasthenia, syphilis with hysteria, or perhaps hysteria or neurasthenia the direct resultant of the poisonous influence of syphilis or of alcohol; and it is necessary that the clinician should separate phenomena due to the toxemia and those due to hysteria, which is fundamentally a cerebral (psychical) affection.

Among the forms of organic disease with ankle-clonus in which I have seen absolute errors of diagnosis made are brain tumor, hemorrhage or softening of the brain, disseminated sclerosis, so-called lateral sclerosis, ataxic paraplegia, syphilitic spastic paraplegia, transverse myelitis, and caries with pachymeningitis. In some of these cases, when the incorrect diagnosis was made, sufficient time had not elapsed for a clear decision.

It must perhaps be admitted that in one class of grave hysterical cases, marked and persistent ankle-clonus is sometimes present, although even in these cases, as suggested by Gowers, the clonus may be secondary to the condition of contracture, or the myotatic irritability may be due to nutritional changes.

Rhein, at the meeting of the Philadelphia Neurological So-

[26] Seguin, E. C., Opera Minora.

ciety at which the report of Dr. Burr's patient was presented, spoke of a case which seemed from the history to be clearly hysterical, and in which the phenomena, ankle-clonus being among them, disappeared under hypnotic suggestion. The chief symptoms were spasticity in the legs, general tremor when walking, spasmodic strabismus, segmental anesthesia, and a true ankle-clonus, which persisted on one side but was easily exhausted on the other. This case upholds the view that true ankle-clonus may be present in cases with hypertonicity and the diathesis of contracture. An interesting medico-legal case, recently observed, lends confirmation to this view. Traumatic hysteria was certainly, in part at least, the true explanation, and in this case ankle-clonus of the most marked and persistent type was present. This patient had met with an injury in attempting to board a car. Her chief subjective symptoms were great pain in the head and left side, insomnia and general nervousness. Examination showed marked paralysis of the right upper extremity with partial loss of power in the lower, analgesia and thermal anesthesia over the right half of the body and limbs. She was unable to walk, stand erect or (apparently at least) to hold up her head. Both lower extremities showed a frequent tendency to spasticity, and at times she had attacks of severe spasm in them. The knee-jerks were exaggerated, and ankle-clonus was present on both sides, but most marked and persistent on the right, which was the side of the anesthesia and paralysis. This, in very general terms, was the condition of the patient at the time of my first examination in October, 1897. She was last examined by me November 11, 1898, when I found present most of the conditions just stated. She was still partially paralyzed in both the upper and lower extremities on the right side, and sensation was still partially lost over a large portion of the same side of the body. The right leg tended to rotate outward at the hip, and she had some contracture both in the upper and lower extremities. Knee-jerks were still exaggerated, but ankle-clonus was absent, although the left leg became spastic when handled. In addition to the hysteria, a hemorrhagic or other lesion affecting the motor tracts may have been present, although the case was undoubtedly in large part one of hysteria.

In hospital and private practice and in medico-legal work, I have examined a very large number of cases of the kind usually classed under the head of hysteria, hystero-neurasthenia, or neurasthenia. Where the diagnosis has been clear, that is, when the existence of an organic lesion could with confidence be excluded, ankle-clonus of any type has been very rarely present, and the persistent form of ankle-clonus has always been absent—excluding from this class only a very few cases in which hypertonicity or the diathesis of contracture was present. I have examined for this point thirty medico-legal cases. In seventeen of these cases the diagnosis of traumatic hysteria or traumatic hystero-neurasthenia, and in thirteen that of traumatic neurasthenia was made. These cases were all examined for muscle-jerks, knee-jerks and ankle-clonus. In only three of the thirty was ankle-clonus of any type present, and in these it was of the abortive or short lived form; in one of the three it was of the spurious type.

In every record of a case presumably hysterical, in which it is stated that foot-clonus is present, a statement should be appended describing the type of clonus. The cases will be few, and the clonus will be of the abortive or spurious form, excepting from this statement a few cases with spasticity and contracture.

PATELLAR CLONUS.

In examining for patellar clonus one of the best methods of grasping and pushing down the patella is by means of the separated thumb and index finger, either using steady pressure or supplementing this with occasional strong pushing movements.

As I sometimes found it difficult to elicit patellar clonus, even when it was present and persistent, owing to the difficulty in keeping up a steady traction and at the same time supplementing this with light percussions, I have had made a simple instrument which might be called the *patella-tractor*, to assist in studying this clinical phenomenon. It consists of an oval-shaped, closed metallic ring with a shank, the whole being attached to a handle which affords an easy grasp. The upper segment of the metallic ellipse is covered with rubber slightly roughened on its inner edge so as not to so readily slip. In taking patellar clonus this ring of metal is slipped over the

patella, the handle of the instrument being grasped below the knee in such a manner that both downward traction and some pressure can be exerted. By pulling steadily downward on the patella tension can without difficulty be kept up indefinitely. (Fig. VII.)

For patellar clonus I examined in all one hundred cases. These cases were chiefly patients in the wards for nervous diseases of the Philadelphia Hospital and included the following diseases:

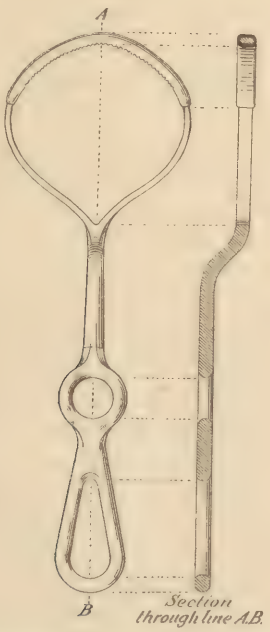


FIGURE VII. Patella-tractor.

Epilepsy, 10 cases; hemiplegia and monoplegia (from hemorrhage, thrombosis or embolism), 19 cases; cerebral syphilis, 4 cases; cerebrospinal syphilis, 3 cases; multiple cerebrospinal sclerosis, 5 cases; paralysis agitans, 4 cases; uremia, 1 case; cerebellar tumor, 1 case; chorea, 1 case; Ménière's disease, 1 case; chronic myelitis, 3 cases; syringomyelia, 1 case; progressive muscular atrophy, 3 cases; senility, 2 cases; functional tremor, 1 case; amyotrophic lateral sclerosis, 2 cases; ataxic paraplegia, 1 case; Friedreich's ataxia, 1 case; spastic paraplegia (probably syphilitic), 2 cases; multiple neuritis, 5 cases; tabes, 28 cases; general paralysis of the insane, 1 case; lead poisoning, 1 case; total, 100 cases.

Out of one hundred cases examined patellar clonus could be elicited in only seven cases. In two of these cases it was present, but only slightly marked; in one case, an old hemiplegic, it was slightly present on the paralyzed side; in the other, a case of senility probably with lacunæ of softening both in the brain and cord, it was slightly present on both sides. In a case of syringomyelia it was well marked on both sides. It was also well marked in two cases of cerebro-spinal syphilis. In one of these cases some paralysis was present in all four extremities, but most decided in the right arm. The patient had had no less than five paretic or paralytic attacks, and a history of syphilis six years before admission to the hospital. The lesions were probably of the nature of diffuse gummatous meningitis or meningomyelitis. Quadriceps-jerk, knee-jerk, gastrocnemius-jerk, ankle-jerk and the front-tap phenomenon were all plus. Patellar clonus was present on both sides, and the case was interesting because of the fact that while patellar clonus was present ankle-clonus was absent. In another case of cerebrospinal syphilis with well marked cerebral, cranial-nerve and spinal symptoms patellar clonus was present on the right side, but on the left was unobtainable, apparently because of strong contractures in the flexor muscles of the thigh. In this case also the quadriceps-jerk, knee-jerk, gastrocnemius-jerk and ankle-jerk and front-tap phenomenon were all present, but ankle-clonus was absent, although patellar clonus could be elicited on the right side. These two cases show that patellar clonus may be present in the absence of ankle-clonus, a clinical fact which would seem to call in question the statement that has been made that patellar clonus represents the highest grade of reflex excitability of muscle or tendon in the lower extremities. Ankle-clonus may be present and patellar clonus absent, and this is probably the most frequent clinical arrangement of these two phenomena; but patellar clonus may be present, as just shown, when ankle-clonus is absent. Reference has already been made to one case of multiple neuritis with symptoms of erythromelalgia, in which slight patellar clonus on one side was elicited at one of the examinations.

A study of the deep reflexes of the lower extremities, including patellar clonus, proved to be interesting in a case of

acute uremia admitted to the nervous wards of the Philadelphia Hospital during the time that I was engaged in examining the patients for most of the facts contained in this paper. This patient, J. S., 54 years old, was admitted to the hospital December 18, 1898. When admitted he was in a semi-conscious state; his pupils were contracted and did not react to light. His pulse was 50, fairly full and slightly irregular; temperature 99.04 F.; respiration 28; his right arm was paretic; both legs were spastic. His urine was one-sixth albumin and contained granular casts. A study of the deep reflexes of the lower extremities shortly after admission showed quadriceps-jerk on both sides marked, knee-jerk much exaggerated, especially on the right; gastrocnemius-jerk present; ankle-jerk present on both sides, but more marked on the right than on the left, a tap on the right tendo Achillis causing slight clonus; on the right ankle-clonus was present but not very persistent, on the left it was absent; patellar clonus was marked on both sides. Examinations on admission were made by my interne, Dr. Merritt. Twenty-four hours later, after the patient had been on active treatment for uremia, which consisted chiefly of hot packs, elixir of gloinoin and Bascham's mixture, all the tendon and muscle phenomena, including patellar clonus, were present, but were less marked than on admission. It is of passing interest to note in this case as in the last two mentioned that while patellar clonus was present on both sides, ankle-clonus could be elicited only on one.

Since taking up this subject anew I have not had the opportunity of examining any considerable number of functional affections for patellar clonus, but on analogy and on theoretical grounds I would be inclined not to expect patellar clonus in hysteria, neurasthenia and other functional nerve disorders, for the same reasons that I have given in stating my views with regard to the significance of ankle-clonus in the differentiation of organic from functional disease. It is not improbable that patellar clonus will be found in those cases of hysteria in which hypertonicity and the diathesis of contracture are present—cases of spastic and convulsive hysteria; cases of tetany, and some cases with choreic and athetoid phenomena.

v. Bechterew[27] says that patellar clonus is very common

[27] Bechterew, W., Neurologisches Centralblatt, 1895, p. 1157.

in cases of myoclonus multiplex, not infrequently the standing position alone being sufficient to throw the muscles of the anterior part of the thigh into clonic contraction.

TENDO-ACHILLIS JERK IN TABETICS.

Babinski,[28] before the Société Médical des Hospitaux de Paris, at the meeting held October 21, 1898, stated that in tabes most frequently the knee-jerk and the tendo-Achillis jerk are both abolished. Sometimes the alterations or disorders in the reflexes are on one side only, but the phenomena may be crossed. In rare cases the knee-jerk is abolished, and that of the tendo Achillis is normal. Finally in some cases the knee-jerk is normal, and that of the tendo Achillis is lost or impaired. Babinski thinks that the involvement or abolition of the reflex of the tendo Achillis is a sign as important in tabes as the sign of Westphal. He believes also that this reflex should be systematically examined, and that it is probable that in any of the cases of tabes where the patellar reflex is preserved, one can establish alteration of the reflex of the tendo Achillis.

Stimulated by this brief communication of Babinski, I have recently examined twenty-eight cases of tabes and tabulated the results. These results were, however, so uniform in character that they can be expressed in a few words. The ankle-jerk (tendo-Achillis jerk) was present in only three cases out of the twenty-eight. In one case the Achillis-jerk was quite marked on both sides. In this patient ataxia was present in both the lower and upper extremities. In a second case the Achillis-jerk was present but slight on both sides. In a third case the Achillis-jerk was not present except by reinforcement, when it could be brought out on the right side only. These three cases were all typical illustrations of tabes from three to nine years' standing, with sensory, cranial-nerve, and other well known phenomena. The quadriceps-jerk, knee-jerk, and gastrocnemius-jerk were present in all, and in one of the cases in which the Achillis-jerk was present the gastrocnemius-jerk was distinctly plus. Besides the twenty-eight cases of tabes, the other seventy-two cases in which patellar clonus was studied

[28] Babinski, Le Progrés Médical, 3e serie, T. 8. No. 44, Oct. 29, 1898, p. 301.

were also examined for tendo-Achillis jerk, and it was present on both sides in all cases.

In one case of tabes the study of the tendo-Achillis jerk was of decided value in reaching a correct diagnosis. This was a case sent to me for opinion by two physicians. The diagnosis of neurasthenia had been given, and the existence of tabes was regarded as in doubt, chiefly for the reason that both knee-jerks were present, although the patient had a recent history of what appeared to be shooting pains and some evidences of probable slight involvement of the bulbar nuclei. Ataxia was not present, but the tendo-Achillis jerk was lost on both sides —quadriceps-jerk, knee-jerk, and gastrocnemius-jerk being present. I concluded that the case was organic, probably a somewhat irregular form of tabes.

A study of the tendo-Achillis jerk may, therefore, be of considerable value in clinching the diagnosis in a doubtful case, and especially in a case in which the region of the patellar reflex arc in the cord has, up to the time of the examination, escaped in whole or in part. My study of the subject shows that the sacral cord is usually involved in cases of well marked tabes; but that the degeneration may in rare instances be confined for a considerable time to the sacral nerves and sacral portion of the cord.

Recently in a case of tumor of the cerebellum—a case presented to the Philadelphia Neurological Society, December 19, 1898, by Dr. William G. Spiller, examined in consultation by the writer, and operated upon later by Dr. John B. Roberts, a careful study was made of all the tendon and muscle phenomena. All the phenomena with which we are concerned in the present communication, quadriceps-jerk, knee-jerk, gastrocnemius-jerk and ankle-jerk were absent, as were also front-tap, ankle-clonus, and patellar clonus. A superficial examination of this case might have led to the diagnosis of tabes, and the case is of interest as pointing to one use to which the study of the muscle and tendon phenomena of the lower extremities in the differential diagnosis of encephalic tumor from tabes may be turned. When the *muscle* as well as the tendon phenomena are absent the diagnosis of tumor is more probable. What is the cause of the absence of these phenom-

ena in tumor cases? While their absence may be explicable on the theory of cerebellar influence, they are in some instances better explained on the theory of a more or less general toxemia excited by the growth. This toxic influence is, perhaps, in the first place exerted on the posterior roots of the spinal cord. It is well known that degeneration of the posterior spinal roots has been found in a certain percentage of cases of brain tumor. In all the twenty-eight cases of tabes studied during the preparation of this paper, both the quadriceps and gastrocnemius (muscle) phenomena were present, although in some of these it was apparently below the normal. Why should the muscle as well as the tendon phenomena be absent in these cases of brain tumor without direct involvement of the spinal cord and posterior nerve roots, while in the vast majority of cases of tabes the muscle response is preserved? It has occurred to me that the microscopical findings in the first case recorded in this paper may have some bearings on the explanation of this fact. It will be recalled that both muscle and nerve disease were distinctly present, and in fact the evidence of muscle degeneration predominated. The toxic influence exerted directly on the muscle may be the proper solution of the problem.